D5 34. (amended) The method according to claim 14, wherein an increased level of pepsinogen I concentration is indicative of a corpus gastritis.

REMARKS

This is in response to the Official Action mailed August 26, 2002 for the above-captioned application. Applicants request an extension of time sufficient to make this paper timely and enclose the appropriate fee.

Reconsideration of the application, as amended, in view of the remarks herein is respectfully requested.

The Examiner objected to the drawings submitted. With respect to the submission of new versions of the original drawings, Applicants have indicated that these will be submitted upon receipt of an indication of allowance, and it is believed that this is appropriate procedure given the filing date of this application.

Claims 14-43 are pending.

The Examiner rejected claims 14-38 under 35 USC § 102(b) as being anticipated by Lindgren, et al. Applicants respectfully traverse this rejection. Lindgren does not disclose a method for diagnosing gastritis in a human by evaluating a blood sample for the presence of antibodies specific for H,K-ATPase, Helicobacter pylori, **and** the concentration of pepsinogen I and comparing these to what is found in a normal population to arrive at a diagnostic result as required in amended claim 14. Lindgren merely compares the tests to see which one works best. There is no consideration of tests results in combination, and there is no teaching or suggestion that diagnosis for a single sample would or should be determined based on the results of more than one of the tests. The Examiner's statement that "since the methods appear to be the same as the claimed methods, then **inherently** one would also the results of the tests in combination to classify the condition" is not supported by the reference. Indeed, the conclusion of the Lindgren reference is that "serum pepsinogen A is superior to H+,K+-ATPase antibodies aand the Schiiling test." (Lindgren, Page 587) Furthermore, the Examiner's argument is legally incorrect, since anticipation on the basis of inherency requires that the assumed teaching of the reference is

a necessary result of the actual teaching, not merely a possible result. Thus, the anticipation rejection of claim 14, as amended, and all of the claims dependent thereon should be withdrawn.

With respect to the dependent claims which are also rejected as anticipated, Applicants note that the Examiner has commented on only two of these, claims 19 and 20, to provide an assertion that Lindgren teaches this aspect of the invention. Applicants point out, however, that claims 19 and 20 (as well as other claims) both dependent from claim 16, and that the Examiner has not indicated how claim 16 (or claim 31 which contains the same limitation) could be deemed to be anticipated by the Lindgren reference. There is no teaching in this reference of multiplying two test results together to generate a number that serves as a further diagnostic indicator. Thus, claim 16 also is not anticipated for this separate reason, and neither are claim 31 nor the claims dependent on claim 16 (claims 17-23).

Furthermore, with respect to claim 19, this claim states that an increased level of pepsinogen I concentration is indicative of corpus gastritis. This limitation is also found in claim 34. The pages which the Examiner cites in Lindgren as teaching the elements of this claims do not even mention pepsinogen I. Thus, the basis for the assertion that claim 19 is anticipated by Lindgren is unclear.

Applicants respectfully submit that an anticipation rejection must show where the reference teaches the limitations of the claim rejected, and that the Examiner has therefore failed to present a procedurally complete rejection with respect to any claim but claim 14. This rejection, however, is factually incorrect as shown above. Thus, the anticipation rejection based on Lindgren should be withdrawn.

The Examiner rejected claims 39-43 under 35 USC § 103(a) as being obvious over Lindgren, et al. Applicants respectfully traverse this rejection. The examiner has still not provided any reasons to make the kit which he alleges are obvious. Applicants are not seeking to claim reagents for any of the test performed individually. They only seek to claim a kit which is assembled for convenient use in the method of Applicants invention, a method which is not taught by Lindgren. One laboratory performing a research comparison of several tests to see which works best, research which need never be repeated, cannot be reasonably said to provide

motivation a kit so that it would be convenient for others to repeat the work. It is also noted that the Examiner makes certain observations as to what is "well known in the art", and relies on these rather than a reference to support his position. For example, the Examiner states that "supplying three immunoassay indicators in the form of a kit comprising reagents suitable for the well known indicators ... are well known in the art." (Office Action, Page 6). Such reliance is legally improper, and the Examiner must supply a reference or a sworn declaration of personal knowledge detailing that which is "well-known," or must withdraw the rejection. *In re Ahlert*, 165 USPQ 418, 420-21 (CCPA 1970).

The Examiner rejected claims 14-43 under 35 USC § 103(a) as being obvious over Oksanen, et al. in view of Ma J.Y., et al. Applicants respectfully traverse this rejection. In this case, the Examiner has found two references which between them teach the three tests recited in claim 14 individually. The basis for this rejection is the Examiner's statement that "the analysis of multiple analytes or more indicators associated with gastritis provides reliable method for screening gastritis." Applicants of course cannot disagree with this statement of reliability, because that is their invention. There is no suggestion in the art, however, that combining the results of the three tests identified by Applicants in their claims would be in any way desirable or would in any way justify the increased cost associated with performing more tests. The Examiner may not freely pick and choose through the literature using the invention as a guide to arrive at a rejection for obviousness. That, however, is what has been done here. Furthermore, it is noted that the Examiner has again lumped all of the claims together rather than addressing why the independent claims would be suggested by the art. Thus, he has failed to present a *prima facie* case of obviousness.

The Examiner rejected claims 19, 20, 21, 26, 27, 34 and 35 under 35 USC § 112, first paragraph, as containing subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the art that the inventor had possession of the claimed invention at the time the application was filed. The Examiner states that these claims

lack support in the application as filed. Applicants respectfully disagree.

Claims 19, 26 and 34 state that an increased level of pepsinogen I concentration is indicative of corpus gastritis. The reference to "optionally without any autoimmunity involved" has been deleted. The relationship between elevated levels of pepsinogen I and corpus gatsrtis is reflected in the PGI data for Group 4.

Claim 20, 27 and 35 refer to the relationship between H+-K+-ATPase antibodies and autoimmune corpus atrophy. The Examiner has not specifically stated why this claim is objectionable, but assume it is because the word "autoimmine" is not found in the original application. Applicant point out, however, that the Examiner has cited art (Lindgren) which refers to the automimmune nature of atrophic gastritis. The specification speaks of the relationship between atrophic gastritis and H+K+-ATPase antibodies. The substitution of one art recognized term for another with the same meaning is not new matter.

With respect to claim 21, Applicants point out that 88% of serological group 2A-D had antral gastritis (antrum) or pangastritis and were observed to be H pylori positive. (Page 10, lines 8-11.

The Examiner rejected claims 14-43 under 35 USC § 112, second paragraph, as being indefinite.

The Examiner stated that Claim 14 contained "an improper Markush group." While noting that there is nothing per se objectionable to the use of the term "or", this word does not appear in claim 14. Thus, the basis for this rejection is unclear. Nevertheless, claim 14 has been amended to clarify the phrasing of that claim.

With respect to the phrase "diagnosing possible presence of gastritis" this is meant to reflect the fact that the scope of the claim does not depend on the result of the test. A patient may present with symptoms suggesting gastritis, but produce test results such that gastritis is not in fact diagnosed. Thus, the person tested has only a possible presence of gastritis.

With respect to the term "altered levels" this is defined on page 9 of the specification as levels either significantly above or significantly below a normal control.

Claim 15 has been amended to correct the antecedent basis problem.

The Examiner states that the phrase "concentration of pepsinogen I" or "pepsinogen I concentration" are unclear, rendering claims 14, 18, 22, 25, 26, 29, 33 34, 37, 38 and 43 indefinite, but has not explained the rejection. The phrases have their ordinary meaning: the amount of pepsinogen I in a unit volume of sample.

The Examiner rejected claims 16 and 31 saying it broadened claim 14. This is erroenous for two reasons. First, the asserted Markush language in claim 14 did not exist. The claim lists three tests, all of which must be performed. Second, even if claim 14 did contain Markush language, the method of claim 14 was still in terms of "comprising" language and did not preclude additional steps. Since claim 16 requires an additional step, based on a combination of the result from the three recited assays, one could infringe claim 14 without infringing claim 16. Thus, the rejection of claim 16 on this ground is in error.

While there is nothing indefinite in the term "optionally" the rejection of claims 19, 26 and 34 on this ground is rendered moot as these claims have been amended to remove the term.

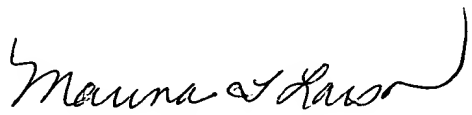
Claim 39 is said to be indefinite in the recitation of "reagents suitable for detecting antibodies". The Examiner has not said why a person skilled in the art would fail to understand this language, but instead asks "what reagents suitable for detecting antibodies?" The burden is on the Examiner in the first instance to show how the scope of the claim is unclear, and may not meet this burden simply by saying that the claim is broad, or that specific reagents are not listed. Thus, the rejection of claim 39 fails as a matter of law.

The Examiner rejected kits claims 39-43 under 35 USC § 103(a) as being obvious over Lindgren, et al. in view of Harkonen. Applicants respectfully traverse this rejection. As noted above, there is no reason in Lindgren to make a kit containing several sets of test reagents, because the purpose of Lindgren was to find which **one** test performed best. Furthermore, the Examiner chooses to pluck H+K+-ATPase antibodies detection out of Lindgren and graft it into Harkonen but provides no reason (other than hindsight to support a rejection) to do so. He has not indicated any reason why the two tests would achieve the same result, nor any reaosn why one would replace an apparently successful test with one that Lindgren says does not work very

well. (Page 586, 3rd full paragraph). Thus, the Examiner has failed to put forward a *prima facie* obviousness rejection.

For the foregoing reasons, Applicants submit that the claims of this application are in form for allowance. Favorable reconsideration and allowance of all claims are respectfully urged.

Respectfully submitted,



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MARKED UP CLAIMS SHOWING CHANGES

14. (amended) A method for diagnosing possible presence of gastritis in a human by evaluating a blood sample comprising the steps of:

assaying the blood sample for the presence of antibodies specific for H,K-ATPase,

assaying the blood sample for the presence of antibodies specific for Helicobacter pylori, [and]

assaying the blood sample for the concentration of pepsinogen I, and

comparing [whereby] the presence of H,K-ATPase antibodies, Helicobacter pylori antibodies, and pepsinogen I concentration [are compared between themselves and in relation] to the respective values of H,K-ATPase antibodies, Helicobacter pylori antibodies, and pepsinogen I concentration of a normal population,

and rendering a diagnosis of gastritis when [wherein altered] levels of H,K-ATPase antibodies, Helicobacter pylori antibodies, and pepsinogen I concentration in the sample are detected that are altered as compared to the respective values in the normal population.

15. (amended) The method according to claim 14, wherein the step of determining the levels of H,K-ATPase antibodies, Helicobacter pylori antibodies, and pepsinogen I [said indicators comprising] comprises performing immunoassays for detecting [the indicators] H,K-ATPase antibodies, Helicobacter pylori antibodies, and pepsinogen I.

16. (amended) The method according to claim 15, further comprising the steps of [determining an additional indicator comprising] multiplying the level of pepsinogen I [multiplied] by the level of Helicobacter pylori antibodies to get a number, and [wherein the level

of this additional indicator is compared] comparing the number to a [standard] number calculated similarly for the normal population and wherein this comparison is included in determining whether to make a diagnosis of gastritis.

19. (amended) The method according to claim 16, wherein an increased level of pepsinogen I concentration is indicative of a corpus gastritis[, optionally] without any autoimmunity involved].

26. (amended) The method according to claim 15, wherein an increased level of pepsinogen I concentration is indicative of a corpus gastritis[, optionally without any autoimmunity involved].

31. (amended) The method according to claim 14, further comprising the steps of [determining an additional indicator comprising] multiplying the level of pepsinogen I [multiplied] by the level of Helicobacter pylori antibodies to get a number, and [wherein the level of this additional indicator is compared] comparing the number to a [standard] number calculated similarly for the normal population and wherein this comparison is included in determining whether to make a diagnosis of gastritis.

34. (amended) The method according to claim 14, wherein an increased level of pepsinogen I concentration is indicative of a corpus gastritis[, optionally without any autoimmunity involved].